IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

Application No.:	09/520,087
Confirmation No.:	1098
Filing Date:	March 7, 2000
Applicant:	Valerie Anne SCOTT et al.
Group Art Unit:	2859
Examiner:	Yaritza GUADALUPE
Title:	OPTICAL SIGHT
Attorney Docket:	10215-000022/US

Customer Service Window
Randolph Building
401 Dulany Street
Alexandria, VA 22314
Mail Stop PETITION

June 17, 2009

PETITION TO REVIVE UNDER 37 CFR § 1.137(b)

Sir:

Applicants petition for revival of the above-identified application on the ground that the application was unintentionally abandoned by failing to respond to the May 12, 2004 Office Action by November 12, 2004. Based upon information and belief, the undersigned attorney states that the abandonment was unintentional, and also states that the entire delay in filing the required reply from the due date for the reply until the filing of this petition was unintentional.

To show that the entire delay was unintentional, the following three Statements are concurrently submitted in connection with this Petition.

1. A Statement by Dr. Boman Axelsson (including EXHIBITS A-F), which discusses the cause of the delay in reply that originally resulted in the abandonment up to January 27, 2005;
2. A Statement by Mr. Steven S. Payne (including EXHIBITS G-N), which discusses the cause of the delay from January 27, 2005 to March 13, 2009; and
3. A Statement by Mr. Herman R. Heflin III, which discusses the cause of the delay from March 13, 2009, until the filing of this Petition.

Applicants concurrently submit the reply to the May 12, 2004 Office Action.

Applicants also concurrently submit the Petition fee of $1,620.00 under 37 CFR § 1.17(m). Please charge any additional fees or credit any overpayment to Deposit Account No. 50-4446.

Respectfully submitted,

Herman R. Heflin III

Herman R. Heflin III, Reg. No. 41,060
P.O. Box 1210
Vienna, VA 22183
(888) 703-1110

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

Application No.:	09/520,087
Confirmation No.:	1098
Filing Date:	March 7, 2000
Applicant:	Valerie Anne SCOTT et al.
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Title:	OPTICAL SIGHT
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Alexandria, VA 22314
Mail Stop PETITION

June 17, 2009

STATEMENT BY DR. BOMAN AXELSSON

Sir:

In support of the Petition to Revive Under 37 CFR § 1.137(b) submitted concurrently herewith, please consider the following information.

1. I, Boman Axelsson, am a Patent Attorney at Ström & Gulliksson AB ("S&G"), which is the Swedish law firm responsible for instructing associates regarding the prosecution of United States Application No. 09/520,087 ("the '087 application").

2. On June 14, 2004, James Ray & Associates ("JRA") forwarded an Office Action dated May 12, 2004, to S&G and requested instructions on responding to the Office Action. See EXHIBIT A.

3. On October 6, 2004, S&G sent a report by facsimile to JRA, including instructions on responding to the May 12, 2004 Office Action. The report instructed JRA to prepare and file a response by October 12, 2004, which is more than one month prior to the final due date of November 12, 2004. See EXHIBIT B.

4. On December 22, 2004, and January 12 and 13, 2005, S&G sent reports by facsimile to JRA, requesting JRA to confirm that a reply to the May 12, 2004 Office had been filed, and to send a copy of the filed reply to S&G. See EXHIBIT C.

5. On January 12, 2005, JRA sent a report to S&G, indicating that JRA never received instructions on how to proceed with the May 12, 2004 Office Action, and that a Notice of Abandonment had been received in the '087 application. See EXHIBIT D.

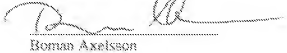
6. On January 26, 2005, S&G sent a report by facsimile to JRA, including instructions to immediately file a petition to revive the '087 application. See EXHIBIT E.

7. As early as January 27, 2005, S&G contacted Steven S. Payne to initiate a transfer of the '087 application from JRA to Steven S. Payne. See EXHIBIT F.

8. The correspondence labeled EXHIBITS A-F were either sent by or directed to Tore Ström, who is now retired from S&G.

9. I declare that all statements made herein of my own knowledge are true, and that all statements made on information and belief are believed to be true. These statements were made with knowledge that willful false statements and the like so made are punishable by fine or imprisonment, or both, under Section 1001 of Title 18 of the United States Code, and that such willful false statements may jeopardize the validity of the application or any patent issuing thereon.

Respectfully submitted,



Boman Axelsson

P.O. Box 4188
Malmö, Sweden
SE-203 13
011 46 40 757 45

EXHIBIT A

James Ray & Associates

James O. Ray, Jr. *

Amos Bartoli *
Michele K. Yoder *
Robert D. Latti
Alexander Pokot *

* reg US Patent Agent
+ admitted to TX Bar

2640 Pitcairn Road
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E-mail: jray@jrayassoc.com
Telephone: 412-380-0725
Facsimile: 412-380-0748

Of Counsel
Alfred D. Lobo
admitted to practice CO & CAFC Bar

Foreign Patent Manager
Todd A. Ray

June 14, 2004

STRÖM & GULLIKSSON AB
P.O. BOX 4188
S-203 13 MALMÖ
SWEDEN

ATTN: TORE STRÖM

RE: US PATENT APPLICATION NO. 09/520,067
"OPTICAL SIGHT"
YOUR REFERENCE: P 576-040 US
OUR REFERENCE: SG 90428

Dear Mr. Ström:

Enclosed herewith is a copy of the Non-Final Office Action issued by the U.S. Patent and Trademark Office. The deadline for responding to this Office Action is August 12, 2004. If necessary, this deadline may be extended up to three months with the payment of extension of time fees.

In view of the arguments presented in the Office Action Dated February 16, 2004, the Examiner has withdrawn her rejection of claims 15-21 over the teachings of Wiklund (US 3,969,356) in view of Matthews et al (US 4,313,273).

The Examiner has now applied a new grounds of rejection. Claims 15-21 are being rejected under 35 USC 102(e) as being anticipated by the teachings of Hines et al (US 5,935,224). It is the Examiner's position that Hines teaches each and every limitation of the claims. This rejection can be overcome with arguments and/or amendments.

Also, please note that Hines has an issue date which is less than one year prior to the filing of the present application. Thus, Hines can be overcome with affidavit evidence showing prior invention, however the effective filing date of Hines is May 9, 1994. Affidavit evidence must be able to prove invention prior to this date to overcome the reference.

The Examiner also rejects claim 15 under 35 USC 102(b) as being anticipated by Kay et al (US 5,594,584). The Examiner believes that Kay et al also teach each and every limitation of claim 15. This rejection can also be overcome by arguments and/or amendments.

Please instruct us as to how you wish to respond to the Examiner's rejection. Since this office action is non-final, we are free to present any arguments and/or claim amendments in the response.

Please feel free to contact us if you have any questions concerning this communication. I will look forward to receiving your instructions well before the August 12, 2004 deadline.

Sincerely,

Michele K. Yoder

Michele K. Yoder

Enclosures

EXHIBIT B

OCT 0 6 2004

TELEFAX

Ström & Gullikssson
intellectual property consulting

To Company	**James Ray & Associates**
Attn. Name	**Ms. Michele K Yoder**
Telefax	0014123000748
Your ref	SG99428
Pages (total)	3
Date	October 6, 2004
Our ref	P 576-040 US Sm/ms

Ström & Gullikssson IPC AB
P O Box 4188
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Website www.sg.se
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Trust Account NDEASESS ISAN
SE54 3000 0000 0407 3193 6426

RE: DEVELOPMENT
US Patent Application Serial No. 09/520,087

PLEASE CONFIRM SAFE RECEIPT OF INSTRUCTIONS.

Kind regards,
Ström & Gullikssson IPC AB
Sam Sidira

MESSAGE CONFIRMATION

06/10/2004 14:01
ID=STRÖM & GULLIKSSON +46 40237897

DATE	S.P-TIME	DISTANT STATION ID	MODE	PAGES	RESULT	
06/10	00'52"	4123800748	TX	003	OK	0000

06/10/2004 14:00 STRÖM & GULLIKSSON +46 40237897 → 0014123800748 NO.839 D001

TELEFAX

Ström & Gullikssonintellectual property consulting

To

Company **James Ray & Associates**

Name **Ms. Michele K Yoder**

Telefax 0014123800748 Date October 6, 2004

Your ref SG99428 Our ref P S76-040 US Sm/ms

Ström & Gullikssson IPC AB
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Website www.sg.se
VAT No SE556102270701

TELEFAX

Ström & Gullikssson

Intelligent & Creative Consulting

To

Company	**James Ray & Associates**		
Name	**Ms. Michele K Yoder**		
Telefax	0014123800748	Date	October 6, 2004
Your ref	SG99428	Our ref	P 576-040 US Sm/ms
Pages (total)	3		

Ström & Gullikssson IPC AB
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Bank Giro 622-2137
Trust Account NDEASESS IBAN
SE64 3000 0000 0407 3103 5626

GS DEVELOPMENT
US Patent Application Serial No. 09/520,087

PLEASE CONFIRM SAFE RECEIPT OF INSTRUCTIONS.

Kind regards,
Ström & Gullikssson IPC AB

Mona Sedira

James Ray & Associates
2640 Pitcairn Road
MONROEVILLE, PA 15146
USA
Attention: Ms. Michele K. Yoder

Datum 2004-10-06
Er ref SG 99428
Vår ref P 576-040 US

Ström & Gulliksson
intellectual property consulting

Ström & Gulliksson IPC AB
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203 13 Malmö
Besöksadress Studentgatan 1
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Momsregnr SE556102170701
Postgiro 52 27 20-2
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Klientmedel Nordea 4073 103 662-6

GS DEVELOPMENT AB
US Patent Application 09/520,087
OPTICAL SIGHT

Dear Ms. Yoder:

I thank you for your letter of June 14, 2004, and beg you to prepare and file a reply to the pending Office Action in the above-referenced case which is due on October 12, 2004.

The argumentation presented by the Examiner indicates that she has misinterpreted the references applied and I can see no reason for further amendment of the claims.

Hines et al. discloses a distance measurement apparatus, and the part of interest of this apparatus is the view finder 108 which is a dot sighting device including a tube 126 which defines a light channel. A lens 134 with a partially reflecting surface is located at one end of the tube. A light source 124 is located in the tube to produce a light spot on the partially reflecting surface. The light surce can be turned on and off by means of a switch 144. It is not stated that the light source is a laser diode and it is not clearly shown how the light source is connected to an energizing circuit. In any case it cannot be found in Hines that a pulsating electric current is applied to the light source, and there are no control means operatively connected with an energizing circuit for the light source for adjusting an intensity of the light spot generated by the light source on the reflecting surface of the lens 134 by pulse width modulation of the light source. The Examiner refers to the laser diode 120 of the distance measurement apparatus which emits light from the distance measurement apparatus but has nothing to do with the view finder. The description of Hines columns 7 and 8 , lines 51 - 67 and 1 - 6, respectively, has nothing to do with a time out circuit for light source 124. The Examiner's

argumentation regarding Hines in the Office Action is based on a misinterpretation of Hines and can be refuted.

Kay et al. does not relate to a distance measurement apparatus as the Examiner says in the Office Action but to a dot sighting device which in the same way as Hines includes a tube 3 with a light source 14 therein which is described as a red light emitting diode. A light spot is produced on a partially reflecting surface 11 a transparent element at one end of the tube. The diode is not defined as a laser diode. The Examiner maintains that Kay inherently discloses a power source applying a pulsating electric current to the light source. Kay includes no disclosure that could "inherently" be an incentive to the average skilled man to apply a pulsating electric current to the light source or to provide means operatively connected with an energizing circuit for adjusting an intensity of the light spot by pulse width modulation of a laser diode generating the sight spot. Also regarding Kay the Examiner's argumentation can be refuted.

Ström & Gulliksson

Please consider the two citations and add such further arguments as you deem proper considering US patent law and practice.

I look forward to receiving your report together with a copy of the reply to the Office Action as filed.

Sincerely,

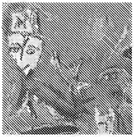
Tore Strom

EXHIBIT C

FACSIMILE 0014123800748

James Ray & Associates
2640 Pitcairn Road
MONROEVILLE, PA 15146
USA
Att: Michele Yoder, Esq.

VIA FACSIMILE
TOTAL 2 SHEETS



EXTREMELY URGENT

January 13, 2005

Date January 12, 2005
Your ref SG99428
Our ref P 576-040 US Sm/ms

Ström & Gulliksson
intellectual property consulting

Ström & Gulliksson AB
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Bank Giro 622-2137
Trust Account NOEASESS IBAN
SE54 3000 0000 0487 3103 6626

GS DEVELOPMENT AB
US Patent Application No. 09/520 087
OPTICAL SIGHT

Dear Ms. Yoder:

With reference to our letter of December 22, 2004 we beg you to send us by return facsimile a copy of the amendment due on October 12, 2004, which we suppose you have filed.

Very truly yours,

Tore Ström

What happened in this ~~copy~~ case?
Our clients want a copy of the amendment
Please reply!

MESSAGE CONFIRMATION

12/01/2005 14:55
ID=STRÖM & GULLIKSSON +46 40237897

DATE	S.R-TIME	DISTANT STATION ID	MODE	PAGES	RESULT	
12/01	00'47"	4123800748	TX	003	OK	P000

12/01/2005 14:54 STRÖM & GULLIKSSON +46 40237897 → 0014123800748 NO.332 P001

FACSIMILE 0014123800748

James Ray & Associates
2640 Pitcairn Road
MONROEVILLE, PA 15146
USA
Att: Michele Yoder, Esq.

EXTREMELY URGENT

Ström & Gullikssons

Date January 12, 2005
Your ref SG99428
Our ref P 576-040 US Sm/ms

Ström & Gullikssson AB
P O Box 4128
SE-203 13 Malmö
Sweden
Visiting Address Studentgatan 1
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E-mail mail@sg.se
Website www.sg.se
VAT No SE556102270701

GS DEVELOPMENT AB
US Patent Application No. 09/520 087
OPTICAL SIGHT

Dear Ms. Yoder:

With reference to our letter of December 22, 2004 we now wish to send you by

MESSAGE CONFIRMATION

13/01/2005 08:26
ID=STPOM & GULLIKSSON +46 40237897

DATE	S.R-TIME	DISTANT STATION ID	MODE	PAGES	RESULT	
13/01	00'37"	4123800748	TX	002	OK	0000

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FACSIMILE 0014123800748

James Ray & Associates
2640 Pitcairn Road
MONROEVILLE, PA 15146
USA
Att: Michele Yoder, Esq.

VIA FACSIMILE
TOTAL 2 SHEETS

EXTREMELY URGENT

January 13, 2005

Date January 12, 2005
Your ref SG9942B
Our ref P 576 040 US Sm/ms

Ström & Gullikssson
intellectual property consulting

Ström & Gullikssson AB
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SE-203 13 Malmö
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Website www.sg.se
VAT No SE556102276701

GS DEVELOPMENT AB
US Patent Application No. 09/520 087
OPTICAL SIGHT

Dear Ms. Yoder:

MESSAGE CONFIRMATION

22/12/2004 11:44
ID=STRÖM & GULLIKSSON +46 40237097

DATE	S.R-TIME	DISTANT STATION ID	MODE	PAGES	RESULT	
22/12	00'22"	4123800748	TX	001	OK	0000

22/12/2004 11:41 STRÖM & GULLIKSSON +46 40237097 → 0014123800748 NO.164 0001

FACSIMILE 0014123800748

URGENT

James Ray & Associates
2640 Pitcairn Road
MONROEVILLE, PA 15146
USA
For the attention of Ms. Michele Yoder

Artist: Björn Kjellmark

Date	December 22, 2004
Your ref	SG99428
Our ref	P 576-040 US Sm/ms

Ström § Gulliksson
Intellectual property consulting

Ström & Gulliksson IPC AB
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Visiting Address Studentgatan 1
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E-mail mail@sg.se
Website www.sg.se
VAT No SE556302230701

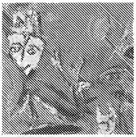
GS DEVELOPMENT AB
US Patent Application Serial No. 09/520087

Dear Ms. Yoder:

We have still not received your report concerning the filing of amendment due

FACSIMILE 0014123800748

URGENT



James Ray & Associates
2640 Pitcairn Road
MONROEVILLE, PA 15146
USA
For the attention of Ms. Michele Yoder

Date: December 22, 2004
Your ref: SG99428
Our ref: P 576-040 US Sm/ms

Ström & Gulliksson

intellectual property consulting

Ström & Gulliksson IPC AB
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Trust Account NDEASESS IBAN
SE54 3000 0000 0407 3103 6626

GS DEVELOPMENT AB
US Patent Application Serial No. 09/520087

Dear Ms. Yoder:

We have still not received your report concerning the filing of amendment due on October 12, 2004. Please send copy of amendment as soon as possible.
PLEASE CONFIRM BY RETURN THAT THE AMENDMENT HAS BEEN FILED.

This case is very urgent.

Very truly yours,
Ström & Gulliksson IPC AB

Tore Ström by Mona Sédira

A subsidiary of the [illegible] Group
represented in Aalborg, Aarhus,
Atlanta, Copenhagen, Gothenburg,
Helsingborg, Linköping, Lund,
Malmö, Munich, Reykjavik,
Sorrnebo, Stockholm

EXHIBIT D

James Ray & Associates

James O. Ray, Jr. `

Amos Bartoli *
Michele K. Yoder `
Robert D. Lotti
Alexander Pokot *

* reg US Patent Agent
` admitted in TX Bar

2640 Pitcairn Road
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E-mail: jray@rayassoc.com
Telephone: 412-380-0725
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Of Counsel
Alfred D. Lobo
admitted to practice OH & CAFC Bar

Foreign Patent Manager
Todd A. Ray

January 12, 2005

RECEIVED
2005 -01- 13
Ström & Gulliksson
Malmö

STROM & GULLIKSSON
P.O. BOX 4188
SE-203 13 MALMO
SWEDEN

ATTN: TORE STROM

RE: U.S. APPLICATION SERIAL NO. 09/520,087
"OPTICAL SIGHT"
YOUR REF: P 576-040 US
OUR REF: SG 99428

Dear Mr. Strom:

In reference to your facsimile dated January 12, 2005 with respect to the Office Action due August 12, 2004 for the above-referenced application.

On June 14, 2004, a copy of the Non-Final Office Action issued by the United States Patent and Trademark Office with a deadline of August 12, 2004 was sent to your office asking you to instruct us with your course of action to the Examiner's rejection.

Again on August 20, 2004 a reminder was faxed to your office. We never received a response from you with instructions on how to proceed with this Office Action. As you are aware we can not proceed without your instructions.

On December 2, 2004 we received a Notice of Abandonment from the United States Patent and Trademark Office for failure to file a proper reply to the Office Action.

However, we can petition to revive the application if necessary.

Please let me know your course of action on this matter.

Sincerely yours,

Michelle K. Yoder

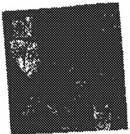
MKY:nf

EXHIBIT E

VIA FACSIMILE
TOTAL 5 SHEETS

FACSIMILE 0014123800748

James Ray & Associates
2640 Pitcairn Road
MONROEVILLE, PA 15146
USA
Ms. Michele Yoder



Ström & Gulliksson
Intellectual property consulting

Date January 26, 2005
Your ref SG 99428
Our ref P 576-040 US Sm/ms

Ström & Gulliksson AB
Box 4188
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Bankgiro 622-2137
Klientmedel Nordea 4073 181 662-6

GS DEVELOPMENT AB
US Patent Application No. 09/520,087
OPTICAL SIGHT

Dear Ms. Yoder,

We refer to your facsimile letter of January 12, 2005.

We acknowledge receipt of the reminder of August 20, 2004 sent from your office.

On October 6, 2004 we sent instructions for answering the Office Action. As we did not receive confirmation of the receipt we sent a reminder on October 10, 2004. On that date we received confirmation of our instructions stamped October 6, 2004 as you can see from the **enclosed copy**. The term for response could be extended until November 12, 2004 but no reminder was received by this office during the period October 10 – November 12.

Why did you not report the Notice of Abandonment of December 2, 2004?

This case is extremely important to our client so of course a petition to revive the application should be filed immediately. Copy of the instructions is sent once again.

Please confirm safe receipt of these instructions by return facsimile.

received
1-26-05

Very truly yours,

Tore Ström

A subsidiary of the AWAPATENT Group
represented in Aalborg, Aarhus, Alicante, Copenhagen, Gothenburg, Helsingborg, Linköping, Lund, Malmo, Munich, Reykjavik, Ronneby, Stockholm

MESSAGE CONFIRMATION

26/01/2005 16:06
ID=STRÖM & GULLIKSSON +46 40237697

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26/01/2005 16:07 STRÖM & GULLIKSSON +46 40237697 → 0014123800749 NO.624 P001

FACSIMILE 0014123800748

VIA FACSIMILE
TOTAL 5 SHEETS

James Ray & Associates
2640 Pitcairn Road
MONROEVILLE, PA 15146
USA

Ms. Michele Yoder

Date January 26, 2005
Your ref SG 99428
Our ref P 576-040 US Sm/ms

Ström & Gulliksson
Intellectual property consulting

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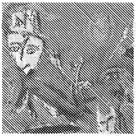
GS DEVELOPMENT AB
US Patent Application No. 09/520,087
OPTICAL SIGHT

Dear Ms. Yoder:

FACSIMILE 0014123800748

VIA FACSIMILE
TOTAL 5 SHEETS

James Ray & Associates
2640 Pitcairn Road
MONROEVILLE, PA 15146
USA
Ms. Michele Yoder



Date	January 26, 2005
Your ref	SG 99428
Our ref	P 576-040 US Sm/ms

Ström & Gulliksson

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203 13 Malmö
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Postgiro 52 37 20-2
Bankgiro 622-2137
Klientmedel Nordea 4073 103 662-6

GS DEVELOPMENT AB
US Patent Application No. 09/520,087
OPTICAL SIGHT

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This case is extremely important to our client so of course a petition to revive the application should be filed immediately. Copy of the instructions is sent once again.

Please confirm safe receipt of these instructions by return facsimile.

Very truly yours,

Tore Strom

EXHIBIT F

Mona Sédira

From: Mona Sédira
Sent: den 1 februari 2005 09:29
To: Steven Payne
Cc: Tore Ström
Subject: RE: Our ref. P 3630-001 US

Hello Steven:

[redacted]

P 0576-040 US
International filing Date March 7, 2000. US filing date March 7, 2000. Inventors are: Valerie Ann Scott, 319 Newmarket Road, CAMBRIDGE CB5 8JE, Alan Edward Green, 33 Devas Bull Way, Milton, CAMBRIDGE CB4 6DF, Euan Morrison, 33 St Philips Road, CAMBRIDGE CB1 3AQ, Great Britain This application has been assigned to GS Development AB, Jägerhillsgatan 15, SE 213 75 MALMÖ, Sweden

If you should need further information please contact me.

With kind regards,
Mona

-----Original Message-----
From: Steven Payne
Sent: den 28 januari 2005 20:28
To: Mona Sédira
Subject: Re: Our ref. P 3630-001 US

Hello Tore and Mona,

Further to my email yesterday, I need the filing dates and the first inventor's name for each of the applications so I can complete the appropriate power of attorney forms. I look forward to your reply. Thanks,

Best regards,
Steve

-----Original Message-----
From: mona.sedira@sg.se
Date: Jan 27, 2005 7:25:57 AM
To: Steven Payne <steven.payne@sg.se>
Subj: Our ref. P 3630-001 US

Hello Steven:

Instruction letter from Tore Ström is enclosed.

Kind regards,
Mona

Ström & Gullikson AB

Malin Sandin
Kanslichef

P O Box 4106
SE-203 13 Malmö, Sweden

Phone +46(0)40 797 45
Fax +46(0)40 23 78 97
E-mail malin.sandin@sq.se
Website www.sq.se

A subsidiary of the ARATOR Group
represented in Denmark, Norway, Sweden, Germany and USA.

PATENT

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

Application No.:	09/520,087
Confirmation No.:	1098
Filing Date:	March 7, 2000
Applicant:	Valerie Anne SCOTT et al.
Group Art Unit:	2859
Examiner:	Yaritza GUADALUPE
Title:	OPTICAL SIGHT
Attorney Docket:	10215-000022/US

Customer Service Window
Randolph Building
401 Dulany Street
Alexandria, VA 22314
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June 17, 2009

STATEMENT BY MR. STEVEN S. PAYNE

Sir:

In support of the Petition to Revive Under 37 CFR § 1.137(b) submitted concurrently herewith, please consider the following information.

1. As early as January 27, 2005, the Applicants' Swedish representative, Ström & Gullikssson AB ("S&G") asked me, Steven S. Payne, to assume responsibility for the prosecution of United States Patent Application No. 09/520,087 ("the '087 application"). I agreed to the representation. See EXHIBIT G.

2. After January 27, 2005, James Ray & Associates ("JRA") forwarded the physical file of the '087 application to me. The physical file that I received did not include the May 12, 2004 Office Action or the November 30, 2004 Notice of Abandonment.

3. On February 17, 2005, S&G asked me to confirm that I would take over responsibility for prosecution of the '087 application, and to report on the status of the '087 application. See EXHIBIT H.

4. On March 21, 2005, I filed (1) a general Power of Attorney (PTO/SB/81), and (2) a Rule 3.73(b) Statement in the USPTO to associate the '087 application with my firm. The Power of Attorney was accepted by the USPTO via a Notice dated April 4, 2005.

5. On June 8, 2005, S&G informed me that it had inquired about the status of the '087 application from JRA, but that JRA had not replied. S&G also instructed me to contact JRA directly to inquire about the status of the '087 application. See EXHIBIT I.

6. On October 20 and November 24, 2005, S&G asked me for the status of the '087 application. See EXHIBIT J.

7. On November 29, 2005, I informed S&G that the '087 application was pending and waiting for the USPTO to act on the application. See EXHIBIT K.

8. I, as a sole practitioner, had the sole right to investigate that status of and revive the '087 application, which was abandoned for failing to respond to the May 12, 2004 Office Action.

9. The reasons for my failure to investigate and file a petition to revive the application in a timely fashion, I believe can be attributed to my mental state during the time in question.

10. In 2003, I was diagnosed by Dr. S. Mark Tanen with a Thyroid disease, specifically Hashimoto's disease. In Hashimoto's, antibodies react against proteins in the Thyroid gland causing gradual destruction of the gland itself and making the gland unable to produce the thyroid hormones the body needs. As a result, I have been taking Synthroid, a thyroid replacement hormone, everyday. Since 2003, my thyroid condition has continued to gradually deteriorate and Dr. Tanen has had to raise my daily dosage of Synthroid several times.

11. It is well known that some of the most profound effects on thyroid hormone imbalance are in the mental arena. Some people with Hashimoto's disease may sleep easily but do not get full refreshment from their sleep. During waking hours, they experience fatigue, apathy and "brain fog" (short-term memory problems and attention deficits). These problems

may affect their daily functioning and cause increased stress and depression. See EXHIBITS L-N for more background on thyroid disorders.

12. The usual treatment for Hashimoto's is taking thyroid hormones in pill form such as the Synthroid I was prescribed.

13. I slowly but steadily started to experience the mental issues such as apathy, brain fog and depression. But since they did not occur quickly but rather developed slowly over time, I did not recognize nor associate these problems with my thyroid disorder. I am also a very private person so I did not share these problems with my family, Dr. Tanen or anyone. I simply maintained a facade that all was well even though some days at work would simply fly by in a fog and nothing would be accomplished.

14. Through 2005-2007, my condition continued to worsen and my depression grew profound. Many work related things were late or missed because of my lack of concentration and the depression I was suffering. Since I am a sole practitioner, there were no colleagues to notice the problems.

15. In January of 2008 and again in April of 2008, I attempted suicide.

16. After the second suicide attempt, I could no longer maintain my façade and my family demanded to know what was happening with me. As a result, I told my family and my clients what had been going on.

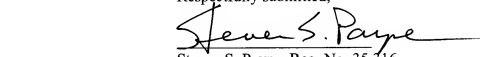
17. I also went to Dr. Tanen to tell him what was happening. After hearing of my condition, he told me that he wished I had come to him and told him about these symptoms when they first started to occur. There are two types of thyroid hormones: L3 and L4. Synthroid is an L4 hormone replacement. While the use of Synthoid is the usual treatment for Hashimoto's disease, some people who are experiencing the same mental problems I was experiencing while just taking Synthroid have found that a combination of L3 and L4 replacement hormones can greatly reduce the mental problems associated with this disease. Doctors are reluctant however to prescribe L3 to patients that are not experiencing the mental problems associated with the disease because L3 hormones can cause damage to the heart. With Dr. Tanen's help, I am now

on a combination of prescription drugs Synthroid (L4) and Cytomel (L3) and I am enjoying a reduction of my symptoms. For brevity I have not gone into the complicated medical theory regarding thyroid disease and the differences between L3 and L4 replacement hormones but have attached EXHIBITS L-N in support for my statements above. I would particularly recommend EXHIBIT L, the article entitled "Use of T3 Thyroid Hormone To Treat Depression" by Dr. Gabe Mirkin.

18. My failure to promptly file a petition to revive the application was unintentional and was due to the mental state I was experiencing during the time in question.

19. I declare that all statements made herein of my own knowledge are true, and that all statements made on information and belief are believed to be true. These statements were made with knowledge that willful false statements and the like so made are punishable by fine or imprisonment, or both, under Section 1001 of Title 18 of the United States Code, and that such willful false statements may jeopardize the validity of the application or any patent issuing thereon.

Respectfully submitted,



Steven S. Payne, Reg. No. 35,316

8027 Iliff Drive
Dunn Loring, VA 22027
(703) 698-1455

EXHIBIT G

Mona Sédira

From: Mona Sédira
Sent: den 1 februari 2006 09:29
To: Steven Payne
Cc: Tore Ström
Subject: RE: Our ref. P 3630-001 US

Hello Steven:

[redacted]

P 0576-040 US
International filing Date March 7, 2000. US filing date March 7, 2000. Inventors are: Valérie Ann Scott, 319 Newmarket Road, CAMBRIDGE CB5 8DE, Alan Edward Green, 33 David Hull Way, Milton, CAMBRIDGE CB4 6DF, Euan Morrison, 33 St Philips Road, CAMBRIDGE CB1 3AQ, Great Britain This application has been assigned to GB Development AB, Jagerhillsgatan 15, SE 213 75 MALMÖ, Sweden

If you should need further information please contact me.

With kind regards,
Mona

-----Original Message-----
From: Steven Payne
Sent: den 28 januari 2005 20:28
To: Mona Sédira
Subject: Re: Our ref. P 3630-001 US

Hello Tore and Mona,

Further to my email yesterday, I need the filing dates and the first inventor's name for each of the applications so I can complete the appropriate power of attorney forms. I look forward to your reply. Thanks,

Best regards,
Steve

-----Original Message-----
From: Mona.Sedira@sg.se
Date: Jan 27, 2005 7:26:37 AM
To: Steven Payne <steven.payne@sg.se>
Subj: Our ref. P 3630-001 US

Hello Steven:

Instruction letter from Tore Ström is enclosed.

Kind regards,
Mona

Hans Sedlis
Paralegal

P O Box 4186
SE-203 13 Malmö, Sweden

Phone +46(0)40 757 45
Fax +46(0)40 23 78 97
E-mail hans.sedlis@aq.se
Website www.aq.se

A subsidiary of the AQ Group
represented in Denmark, Norway, Sweden, Germany and USA.

EXHIBIT H

Mona Sédira

From: Mona Sédira
Sent: den 17 februari 2005 13:57
To: Steven Payne
Subject: Our ref: P 3630-001 US, P 1113-059 US, P 576-040 US (our mail of February 1, 2005)

Hello Steven:

Please confirm by return email that you are now in a position to take over the above-referenced cases.

If no more assistance is necessary for this please take immediately such steps as are necessary for the take over of the cases. When you have taken over the further prosecution of the cases please let us have immedialy a report on the present status of the cases.

Best regards,
Tore Ström/Mona Sédira

Ström & Gulliksson AB

Mona Sédira
Patentegal

P O Box 4188
SE-203 13 Malmö, Sweden

Phone +46(0)40 757 45
Fax +46(0)40 23 78 97
E-mail mona.sedira@sg.se
Website www.sg.se

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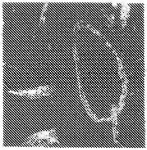
EXHIBIT I

FACSIMILE 0012028289399

Arator IP Law Group
1101 17th St. N.W.
WASHINGTON, DC 20036
USA



Ström & Gulliksson

Date June 6, 2005
Your ref 190-294, 190-299
Our ref P 1113-059 US and P 576-040 US

Ström & Gulliksson AB
P O Box 4188
SE-203 13 Malmö
Sweden
Visiting Address Studentgatan 1
Malmö
Phone +46 (0)40 757 05
Fax +46 (0)40 23 78 97
E-mail main@sg.se
Website www.sg.se
VAT No SE556192276701
Postal Giro 52 27 30-2
Bank Giro 622-2137
Trust Account NDEASESS IBAN
SE94 3000 0000 0487 3183 4626

[redacted]

GS DEVELOPMENT AB
US Patent Application Serial No. 09/520,087

Dear Steve:

We have asked Mr. Ray to give information on the present status of the above-referenced cases which have Mr. Ray's reference numbers SG 99224 and SG99428 but we have not been able to get that information. Also, at telephone calls it has not been possible to speak with Mr. Ray.

I beg you to contact Mr. Ray over the telephone and to repeat the contacts with him until you have got information on the present status of the cases.

Very truly yours,

Tore Ström

EXHIBIT J

Mona Sédira

From: Mona Sédira
Sent: den 20 oktober 2005 11:24
To: 'Steven Payne'
Cc: Tore Ström
Subject: RE: EXTREMELY URGENT
Follow Up Flag: Follow up
Flag Status: Flagged

Dear Steven:

[redacted]

Please send your report concerning status of the other cases. Our ref. P 576-040 [redacted]

[redacted]

Please confirm receipt.

Kind regards,
Mona Sédira

-----Original Message-----
From: Steven Payne [mailto:paynesteven@msn.com]
Sent: den 19 oktober 2005 16:44
To: Mona Sédira
Cc: Tore Ström
Subject: RE: EXTREMELY URGENT

Dear Tore and Mona,

Yes, the due date can be extended for up to 4 months from the October 22, 2005 due date. I have been in contact with the USPTO and they are still trying to determine why this case has not yet been assigned to me instead of James Ray. The other two cases have been transferred to me. If you plan on responding prior to the October 22, 2005 due date, it would be best to respond through James Ray since he is still the attorney of record in the eyes of the USPTO. If I do not hear back from the USPTO by Friday, I will go to the USPTO with copies of the new Power of Attorney and see what can be done.

Best regards,

Steve

From: *Maria Sédira <mona.sedira@sg.se>*
To: *"Steven Payne" <paynesteven@msn.com>*
Subject: *EXTREMELY URGENT*
Date: *Wed, 19 Oct 2005 12:05:28 +0200*
>Our ref: P 3630-001 US Sm/ms
>
>Your ref: 150-253 Our ref: P 3630-001 US

From: Mona Sédira
Sent: den 24 november 2005 13:34
To: Tore Ström
Subject: FW: 150-254 and 150-255

Importance: High

-----Original Message-----
From: Mona Sédira
Sent: den 24 november 2005 13:33
To: 'Steven Payne'
Subject: 150-254 and 150-255
Importance: High

[redacted] US Patent Application No. 09/520,087 OPTICAL SIGHT (GS Development AB)

Hello Steven:

Please inform if the USPTO has registered you as attorney of record for the two above-referenced applications. Furthermore, we kindly ask you to inform about the status of the applications at your earliest convenience.

Kind regards,
Mona

Ström & Gulliksson AB

Mona Sédira
Paralegal

P O Box 4188
SE-203 13 Malmö, Sweden

Phone +46(0)40 257 45
Fax +46(0)40 23 78 97
E-mail mona.sedira@sg.se
Website www.sg.se

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represented in Denmark, Norway, Sweden, Germany and USA.

EXHIBIT K

From: Steven Payne [payne@aratoriplaw.com]
Sent: den 28 november 2005 00:44
To: Mona Sedira
Subject: Re: 150-254 and 150-255

P596-0406

Dear Mona,

Thanks for your email. I have been officially recognized as the attorney of record in both of these cases, [redacted] We are waiting for the USPTO to act on both of these cases. [redacted] No guarantee on when the Examiner in either case will actually act on the case. I will let you know as soon as I hear anything from the USPTO.

Best regards,
Steve

>From: "=?ISO8859-1?Q?Mona_Sedira?=" <mona.sedira@sg.se>
>Date: Thu Nov 24 06:33:15 CST 2005
>To: Steven Payne <payne@aratoriplaw.com>
>Subject: 150-254 and 150-255

>
>[redacted]
>US Patent Application No. 09/526,087 OPTICAL SIGHT (GS Development AB)
>
>Hello Steven:
>
>Please inform if the USPTO has registered you as attorney of record for
>the two above-referenced applications. Furthermore, we kindly ask you
>to inform about the status of the applications at your earliest
>convenience.
>
>Kind regards,
>Mona
>
>
>_____
>
>Ström & Gullikson AB
>
>Mona Sedira
>Paralegal
>
>P O Box 4188
>SE-203 13 Malmö, Sweden
>
>Phone +46(0)40 757 45
>Fax +46(0)40 23 78 97
>E-mail mona.sedira@sg.se
>Website www.sg.se
>
>A subsidiary of the ARATOR Group
>Representing in Denmark, Norway, Sweden, Germany and USA.
>
>NOTE/NOTICE
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>only for use of the individual or entity named above. If you are not the
>intended recipient, or the agent responsible to deliver it to the intended
>recipient, you are hereby notified that any dissemination, distribution or
>copying of this communication is strictly prohibited. If you have received
>this communication in error, please immediately notify us by e-mail, telephone or
>telefax and delete it thereafter from your system.

EXHIBIT L

USE OF T3 THYROID HORMONE TO TREAT DEPRESSION

Gabe Mirkin, M.D.

If you are tired much of the time, your doctor will order blood tests for the two thyroid hormones called T3 and T4 and for the brain hormones called TSH and prolactin. If your TSH is high and your prolactin is normal, you are probably hypothyroid and need to take thyroid hormone to give you more energy and prevent heart and blood vessel damage.

Doctors treat people with low thyroid function with thyroid pills called T4 (Levothroid, one brand name is Synthroid). Many doctors think that a person needs only T4 because the thyroid gland makes T4 and then it is converted to T3 in other tissues. However, some people become depressed when they take just T4 and their depression can be cured when they take both thyroid hormones, T3 and T4.

When a depressed patient comes to me and is taking thyroid hormone, T4, I immediately order a blood test called TSH to check if he or she is getting the correct dose. If the TSH is normal, I reduce the dose of T4 by 50% and add a very low dose of T3 (brand name, Cytomel) because it safer to prescribe too low a dose, rather than too high a dose. Overdoses cause shakiness, irritability, irregular heart beats, clots, and osteoporosis. The patient returns in one month for a blood test, TSH, to see if the total thyroid dose is correct. If the TSH is too high, the thyroid dose is too low and I raise the T3 (Cytomel) dose by 5 to 10 mcg each month until the TSH is normal. Then once a year I check TSH blood levels to make sure that the person's requirements for thyroid hormone are being met.

For example, the usual replacement dose for low thyroid function is 100 micrograms per day. If a depressed patient has a normal TSH, I reduce the T4 dose to 50 mcg/day and add 5 mcg of T3 per day. One month later, if the TSH blood is still too high I raise the T3 dose to 10 or 20 mcg and continue to increase the T3 level each month until the TSH is normal.

Exciting research shows that the thyroid hormone called T3 can help treat depression (1,2,3). Psychotherapy often fails to control depression. Sigmund Freud, the father of psychotherapy, proposed theories about depression, that many psychiatrists do not accept because his writings were his opinions and not presented as scientific data supported by controlled experiments. The dominant theory today is that depression is caused by low brain levels of the neurotransmitters, serotonin and norepinephrine. The drugs such as Paxil, Prozac and Zoloft that treat depression are supposed to raise brain levels of these neurotransmitters. Doctors can also raise brain levels of serotonin by prescribing pills containing T3, a hormone produced by peripheral tissue from T4, which is produced by the thyroid gland. (1) They also prescribe T3 by itself or together with antidepressants. Depression is common among people who have too much or too little thyroid hormone. Doctors usually treat low thyroid function with T4 also known as Levothroid and many people become even more depressed. They treat this depression by prescribing T3 as well as T4.

An article in the Journal of Clinical Psychiatry shows that T3 can be used to treat post traumatic stress disorder, commonly seen in soldiers and people who have been through other causes of terrible emotional trauma (13).

Try to balance T3 and T4 so you will not be taking too much thyroid and harm yourself. 1)If you now take 100 mcg of Levothroid (T4): 2) Lower T4 (Levothroid) to 50 mcg and add Cytomel (T3) 5 mcg each day. 3) One month later, have your doctor draw blood for TSH. 4) If it is normal, you are on the correct dose and should get blood tests TSH once a year. 5) If TSH is too high, increase Cytomel to 10 mcg and hold Levothroid at 50. 6) Draw monthly TSH until it is normal. Keep on raising Cytomel by 5 mcg until TSH is normal.

1) M Weissel.Treatment of psychiatric diseases with thyroid hormones.Acta Medica Austriaca, 1999, Vol 26, Iss 4, pp 129-131.

2) H Meyer, MKH Schafer, K Bauer.Thyrotropin-Releasing Hormone (TRH), a signal peptide of the central nervous system.Acta Medica Austriaca, 1999, Vol 26, Iss 4, pp 119-122.

3) F Konig, C vonHippel, T Petersdorff, W Kaschka.Antithyroid antibodies in depressive diseases.Acta Medica Austriaca, 1999, Vol 26, Iss 4, pp 126-128.

4) A Steiger.Thyroid gland and sleep.Acta Medica Austriaca, 1999, Vol 26, Iss 4, pp 132-133.

5) Jackson IM .Thyroid 1998 Oct;8(10):951-6.

6) Refractory depression: treatment strategies, with particular reference to the thyroid axis. Joffe RT . J Psychiatry Neurosci 1997 Nov;22(5):327-31 .

7) Thyroid hormones in depressive disorders: a reappraisal of clinical utility. Lasser RA , Baldessarini RJ, Consolidated Department of Psychiatry, Harvard Medical School, Boston, Mass., USA. Harv Rev Psychiatry 1997 Mar-Apr;4

(6):291-305

8) The hypothalamic-pituitary-thyroid axis in major depression.Sullivan PF , Wilson DA , Mulder RT , Joyce PR University Department of Psychological Medicine, Christchurch School of Medicine, New Zealand. Acta Psychiatr Scand 1997 May;95(5):370-8

9) S Ramschak Schwarzer, W Radkohl, C Stiegler, NP Dimal, G Leb. Interaction between psychotropic drugs and thyroid hormone metabolism - an overview. Acta Medica Austriaca, 2000, Vol 27, Iss 1, pp 8-10.

10) Dorn et al. Baseline thyroid hormones in depressed and non-depressed pre- and early-puberty boys and girls. J Psychiatry Research 1997(Sept_Oct);31(5):555-67.

11) Birkenhager TK et al. An open study of triiodothyronine augmentation of tricyclic antidepressant in inpatients with refractory depression. Pharmacopsychiatry 1997(Jan);30(1):23-26.

12) SK Rack, EH Makela. Hypothyroidism and depression: A therapeutic challenge. Annals of Pharmacotherapy, 2000, Vol 34, Iss 10, pp 1142-1145.

13)Triiodothyronine augmentation of selective serotonin reuptake inhibitors in posttraumatic stress disorder. O Agid, AY Shalev, B Lerer. Journal of Clinical Psychiatry, 2001, Vol 62, Iss 3, pp 169-173.

Checked 2/8/09

EXHIBIT M

Thyroid Hormone Disorders

(Released May 2001)

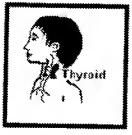
by Jennifer A. Phillips

Review Article

Introduction

Hormones are named from the Greek word *hormon*, meaning "to urge or excite", because they were first discovered to play a role in hunger, sex, flight-or-fight response, and many other basic drives. Hormones serve within the body as invaluable messengers, governors of development, and regulators of metabolism. This Hot Topic will focus on the effects of thyroid hormone (TH) and the disorders that are associated with TH imbalance.

TH, found in all chordate animals, is the only major biochemical molecule known to incorporate iodine, a substance common in the sea but rare on land. Iodine is essential to the structure of TH, and iodine deficiency is the leading cause of hypothyroidism in undeveloped countries. TH is produced by the thyroid, a butterfly-shaped gland behind the larynx, in response to thyroid stimulating hormone (TSH), which is released by the pituitary gland.



TH exists in two major forms. Levothyroxine (T4), with four iodine atoms per molecule, is an inactive form that can be converted into T3, and is produced exclusively by the thyroid gland. Triiodothyronine (T3), with three iodine atoms per molecule, is eight times more effective than T4. It is converted from T4 in the thyroid, brain, liver, and bloodstream, and in various tissues of the body.

The Role of TH in the Body

One important function of TH is helping the body convert food into energy and heat. T3 directly boosts energy metabolism in mitochondria, the powerhouses of cells. T3 triggers rapid protein synthesis and influences mitochondrial gene transcription, the reading of genes and synthesis of proteins from genetic information. These activities cause breakdown of proteins and an increase in free fatty acids, as well as increased oxygen use. TH elevates the heart rate to meet the increased oxygen needs.

TH also regulates body temperature. TSH, which stimulates the thyroid to produce TH, also stimulates brown adipose tissue, a mitochondria-rich tissue, to boost heat production in mammals without muscle activity. TH fluctuates in response to caloric intake and external temperature. During starvation, the body naturally lowers TH, not only to reduce caloric needs, but also to prevent ketone bodies from building up in the blood and kidneys. Ketone build-up, which can also happen in diabetes, can cause damage to the kidneys and other part of the body. Injury and illness lower TH levels, which rebound once the patient is healed.

TH is sensitive to the levels of other hormones besides TSH. Estrogen partially blocks the efficiency of TH, so women compensate by producing more TH than men. This may be why women have larger thyroids than men and are more prone to thyroid disease of all types. Women who take TH replacement pills must increase their TH dosage if they start taking birth control pills, to compensate for the higher levels of estrogen from birth control pills. Growth hormone also partially blocks TH, but it also complements TH in its effects on growth,

development, and metabolism.

TH plays a major role in metamorphosis and development in all vertebrates. It affects development by binding to thyroid hormone receptors (TRs), molecules that then change their shape to an activated form. Once activated by TH, TRs can bind to responsive elements in the DNA, triggering gene transcription. The position of the TR attaching to the responsive elements facilitates the copying of some genes, and blocks others from being copied. Two major forms of thyroid hormone receptors exist: TRα and TRβ.

TRs are nuclear receptors like retinoid A receptors, Vitamin D receptors, and steroid hormone receptors. TRs change configuration when attached to T3, and this changed configuration allows them to attach to responsive elements in the genome. Nuclear receptors are often dimerized (attached to another nuclear receptor of the same or different type), but they remain inactive until bonded by the usual trigger. For example, thyroid hormone receptors dimerized with retinoid X receptors will not activate until they are bonded with T3 or retinoids (derivatives of Vitamin A).

We still do not know all the genes that are regulated by TH. Some TR-responsive elements in the DNA are Alu elements, which are able to move around in the genome on occasion, creating even more Alu elements in the genome. This allows many different genes to come under the control of TH without the genes themselves mutating. Different species may have different genes under control of TH, especially these concerned with development. For instance, while most mammals show similar symptoms of hypothyroidism (fatigue, apathy, etc.), dogs show the additional symptom of seizures. Most chemicals that cause hypothyroidism do not block thyroid receptors in the genes; they only block the efficiency or synthesis of TH. Hence most of our information about which genes are regulated by TH comes from studying genetic disorders in which the TRs are non-functional.

Genetic Disorders Involving TH, TSH, or TRs

Resistance to TH is a genetic disorder caused by mutations in the TRb gene. Patients with this disorder have high TH levels and TSH levels, goiter (enlarged thyroid gland), and mild hypothyroid metabolisms. Clinical effects are less severe than with congenital hypothyroidism and can include short stature, delayed bone maturation, hyperactivity, learning disabilities, and hearing defects, as well as mixed features of hyper- and hypothyroidism. This condition is usually inherited dominantly.

Pendred's Syndrome is caused by a genetic defect that limits the incorporation of iodine into thyroid hormone, which wrecks the structure of the hormone. Pendred's Syndrome can cause hypothyroidism with goiter. The body compensates by producing more TSH and working harder to make enough thyroid hormone that works. The syndrome can also cause more serious problems, such as profound deafness, or non-syndromal deafness alone. These symptoms are present from birth. People who develop hypothyroidism later in life may have ringing in their ears and dulled hearing, but these symptoms are usually correctable by TH therapy, while deafness caused by Pendred's Syndrome is not.

TSH receptor (thyrotropin receptor) gene mutations often cause hyperthyroidism, or TSH insensitivity, which leads to normal TH levels in the blood with elevated TSH levels. TSH has unknown effects on lymphocytes and brain cells; therefore imbalances affecting TSH levels may cause additional, unknown effects on the brain and immune system. One mutation was found in association with Graves' disease. Graves' disease is an autoimmune form of hyperthyroidism, and the genes that seem to increase risk of Graves' disease are associated with immunity.

In humans, thyroid hormone plays a notable role in brain development from the middle of pregnancy to the second year of life. Maternal or fetal hypothyroidism, whether caused by lack of iodine during the pregnancy, or by other problems, can cause a non-genetic condition called cretinism. Babies affected by cretinism can develop normal intelligence if the condition is remedied within a few months, but otherwise they suffer severe, irreversible mental retardation. One severe type of cretinism can also be caused by mutations in the TRa gene, and may be untreatable.

Effects of TH Imbalance: Hypothyroidism

Some of the most profound effects of TH imbalance are in the mental arena. Hypothyroid people sleep easily and do not get full refreshment from their sleep. During waking hours, they

experience fatigue, apathy, and "brain fog" (short-term memory problems and attention deficits). These problems may affect their daily functioning and cause increased stress and depression.

TH acts as a neurotransmitter. TH imbalance can mimic psychiatric disease because T3 influences levels of serotonin, a neurotransmitter integral to moods and behavior. Low levels of T3 can cause depression. Some anti-depressants make hypothyroid patients feel even worse because the medications depress T3 levels. Paradoxically, some substances labelled depressants such as alcohol or opiates can increase T3 levels by impairing the breakdown of T3 in the brain, thus lifting mood. This may be one reason why these substances are so addictive.

Severe hypothyroidism can cause symptoms similar to Alzheimer's disease: memory loss, confusion, slowness, paranoid depression, and in extreme stages, hallucinations. Thyroid disease is one of the many treatable diseases that must be ruled out before arriving at the diagnosis of Alzheimer's, which is incurable and cannot be definitely diagnosed until after death. Risk of hypothyroidism increases with age; by age 60, 17% of women and 9% of men have symptoms of thyroid disease[1] .

Low TH levels also produce fatigue, slight hypoglycemia (low blood sugar), slowed digestion of food, and constipation. Infertility is common. These symptoms can indicate that other diseases are present, particularly because TH levels tend to go down during prolonged illness in an effort to conserve energy. Chronic disease, such as Lyme disease, can mimic (or cause) hypothyroidism. Hypothyroidism is not difficult to diagnose by symptoms, if the patient reports enough symptoms to the doctor and if the doctor thinks of it. Diagnosis can be confirmed by blood tests, but the cause is less easy to discern.

TH imbalance has a profound effect on cardiovascular fitness because TH helps control heart rate and blood pressure. Under hypothyroid conditions, the heart can slow to 30 heart beats a minute and develop arrhythmia. Blood pressure may fall from normal levels of 120/90 to 70/50. Hypothyroidism also weakens muscles, including the diaphragm. As a result, breathing can become less efficient. A goiter impairs breathing even more. Snoring may start or become worse. Fatigue sets in easily; in fact it never quite leaves a person with symptomatic hypothyroidism. Muscles and joints often ache. With respiration impaired and oxygen in short supply, exercise takes a heavy toll on the body, and muscles do not strengthen in response to exercise; nor does stamina improve.

Low thyroid levels actually trigger muscle fibers to change their type, from fast-twitch fibers to slow-twitch fibers. This may be an adaptive strategy for coping with starvation, since blood sugar is low under hypothyroid conditions and fast-twitch muscle fibers require high levels of glucose to operate. Fatty acid levels in the blood are elevated to provide fuel for the fat-burning slow-twitch muscles. However, low oxygen in the blood due to slow heart rate and respiratory problems limits the slow-twitch muscles' effectiveness.

Even after receiving treatment for hypothyroidism, many people find that their caloric needs and ability to handle exercise have changed permanently. Strength training can help restore their fitness, but only after thyroid hormone levels have normalized. Therefore, hypothyroidism affects the ability of people to undergo both aerobic and anaerobic exercise.

Hypothyroidism is the second leading cause of high cholesterol, after diet. When TH levels drop, the liver no longer functions properly and produces excess cholesterol, fatty acids, and triglycerides, which increase the risk of heart disease. High cholesterol may also contribute to the risk of Alzheimer's disease. Hypothyroid patients may develop yellowed skin due to carotenoid (Vitamin A precursors) deposits in the skin when the liver no longer can store enough. Vitamin A usage and synthesis drops as thyroid hormone levels drop.

Effects of TH Imbalance: Hyperthyroidism

Hyperthyroidism is associated with a different set of symptoms. People with this disorder sleep with difficulty and sleep much less than normal. Unlike hypothyroid patients, they exhibit manic-depressive behavior as the TH levels drive their energy levels beyond their physical limits. In fact, thyroid hormone testing is routine at psychiatric admission for suspected manic-depressive patients. Lithium, a common treatment for manic-depression, is known to depress T3 in the brain back to normal levels.

Hyperthyroidism causes accelerated heart rate and fatigue, even when patients are at rest. It

produces lower exercise tolerance because protein and fat catabolism are accelerated, resulting in build-up of ketones. Hyperthyroid people often show a fine tremor in their hands. They have higher resting heart rates, but not higher maximum heart rates for exercise, in comparison to normal subjects. Some experience thyroid storms--high overloads of thyroid hormones that accelerate their heart rate to as high as 300 beats a minute. This is a very life-endangering condition and can result in arrhythmia or heart attack.

Some drugs cause a temporary TH imbalance. Caffeine and other stimulants interfere with T3 and adrenal hormone metabolism while in the body. Smoking depresses TH levels and produces an chronic underlying hypothyroidism as well as low adrenal hormone levels. The hormonal imbalances due to smoking may contribute to the severity of withdrawal symptoms in smokers trying to quit. Research shows that nicotine increases the synthesis of T3 from T4 in the brain, while alcohol and opiates block the breakdown of T3 in the brain[2]. Research into thyroid hormone's role in addiction might lead to better treatment and prevention of drug addiction[3].

Causes of Thyroid Disease

The most common causes of acquired thyroid disorders are iodine deficiency and autoimmune thyroid disease. Iodine deficiency is the major cause of hypothyroidism for much of the world, due to absence of iodine in the diet and/or high consumption of soy, corn, and brassica plants (cabbage, broccoli, brussel sprouts, etc.). These plants produce natural goitrogens. Goitrogens can be largely abolished through proper cooking. In the U.S., salt is iodized to ensure people get enough iodine. Iodine overdose rarely is a problem, as the thyroid gland stores iodine until it is necessary, and releases TH in the less active T4 form, and TH is also bound up by transport proteins in the blood until it is needed. Some experts believe that continual iodine overdoses leads to autoimmune thyroid disease, because it seems to be the major cause of thyroid disorder in developed countries.

Two autoimmune thyroid diseases, Graves' disease and Hashimoto's thyroiditis, are thought to be inherited, but have not been linked positively to any genes. Autoimmune thyroid disease is identified by detecting antibodies in the blood. In the case of Graves' disease, antibodies latch onto an enzyme essential for making T4, and keep it active and continually turned on. Graves' disease is treated by suppressing or killing (removing) the thyroid and then stabilizing the patient on thyroid hormone replacements. In Hashimoto's thyroiditis, antibodies latch onto the same enzyme, but block its function, and help trigger destruction of the thyroid. In the early stages of Hashimoto's thyroiditis, the thyroid may produce too much TH, but as the thyroid is slowly destroyed, the patients TH levels drop. Hashimoto's thyroiditis is treated with thyroid hormone replacements.

Some experts have suggested that autoimmune thyroid disease develops as a result of iodine overconsumption. Both the U.S. and Japan have high levels of iodine consumption and of autoimmune thyroid disease. Japanese people consume iodine because seafood makes up a large proportion of the diet, and Americans do because salt is iodinated and the food industry uses iodine as a machine wash. Other experts believe that pollutants are a more important factor. Pollutant chemicals like polychlorinated biphenyls (PCBs) and dioxins have been shown to interfere with thyroid function and are more prevalent in industrialized countries where thyroid disease levels are high. Autoimmune thyroid disease, either hyperthyroidism or hypothyroidism, is also linked to post-traumatic stress disorder and is often first observed clinically after periods of prolonged stress.

Conclusion

Research on the treatment of thyroid disease is proceeding in promising directions. Autoimmune thyroid disease is being intensively studied, and synthetic antibodies have been produced that neutralize Graves' antibodies in mice. Other studies are uncovering the role of TH in the brain, and finding new genetic causes of thyroid hormone metabolism disorders. TH function is being studied in various vertebrates, and environmental chemicals are undergoing examination as possible TH disruptors. Such research provides hope that autoimmune thyroid disease can one day be attacked at its source.

However, adequate information has not spread into the medical field. Labs performing blood work use overly broad normal ranges of TSH levels. Published research indicates 1-3 µg/ml in the blood (micrograms per milliliter of blood) is the best range of normal[4], but most doctors work under the assumption that values as high as 5.5 are normal, which results in underdiagnosis and undertreatment of many cases of hypothyroidism.

A worse problem is the lack of testing. Though an estimated 200 million people worldwide have thyroid disorders[5], thyroid function tests are rarely given unless the doctor suspects a thyroid disorder, and most doctors do not suspect hypothyroidism in their patients because the symptoms are subtle. Of the estimated 13 million Americans affected by thyroid disease, more than half are unaware of their condition[6]. Thyroid disease affects 8 times as many women as men, possibly because women need higher levels of TH than men do, but it has no age, gender, or ethnic barriers. Patients may have some or all the obvious symptoms: fatigue, lack of focus, depression, constipation, anxiety attacks, dry hair, dry skin, edema (swelling), lack of exercise tolerance, weight gain (especially in the stomach), muscle and joint pains, problems swallowing (due to enlarged thyroid), goiter, facial puffiness, unusual new headaches, loss of eyebrows, lack of sex drive, lowered body temperature, low or high blood pressure, and slowed heart rate. Yet patients may not be diagnosed for years.

The link between high cholesterol and underlying hypothyroidism is vastly overlooked, even though cholesterol's role in heart disease is heavily publicized. People have their cholesterol tested more regularly than their thyroid hormone levels. The result is prescriptions for expensive cholesterol-lowering drugs that don't address the real problem. People diagnosed with high cholesterol, especially those with low body temperature, should have their thyroid function tested before they begin taking such drugs. Also, smokers and other substance abusers should be watched for hypothyroidism (and urged to quit), as stimulants and depressants both can affect TH metabolism.

The under-diagnosis of thyroid disease handicaps research as well as the lives of affected patients. Researchers need to understand the proper function of thyroid hormone and the pathology of thyroid disease to fully understand how our bodies, brains, and immune systems develop and work, in health and in illness. It is impossible to know the prevalence of thyroid disease and figure out all the causes if patients take years on average to be diagnosed. We still do not know what causes the high prevalence of autoimmune thyroid disease in developed countries. Until researchers turn up strong and clear evidence on the cause, more cases of autoimmune thyroid disease will occur every year.

1. Synthroid, Knoll Pharmaceutical Company (http://www.synthroid.com/consumer/1310.htm)
2. Examination of antithyroid effects of smoking products in cultured thyroid follicles: only thiocyanate is a potent antithyroid agent (Acta Endocrinol (Copenh), 1992 Dec, 127(6):520-5)
3. Thyrotropin releasing hormone decreases alcohol intake and preference in rats (Alcohol, 2000 Jan, 20(1):67-91)
4. Weetman AP. Fortnightly review: Hypothyroidism: Screening and subclinical disease. BMJ 1997;314:1175 (19 April) (http://www.bmj.com/cgi/content/full/314/7088/1175)
5. Thyroid Foundation of Canada (http://www.thyroid.ca/Guides/HG00.html)

back to article

6. American Association of Clinical Endocrinologists (http://www.aace.com/pub/spec/tam2001/presstam2001.html)

EXHIBIT N

alt.support.thyroid

T3 Supplementation

The T3 Story

T what?

Let's hear you pronounce it: triiodothyronine. There, now you know why the name is usually shortened to T3. T4 (thyroxine) and T3 are the main thyroid hormones. T3 is five to eight times as strong as T4 (taking into consideration that it's absorbed at a higher rate than T4), and it's biologically more active. T4 is like the food in your refrigerator, while T3 is like the food on your plate.

T4 is slow acting, with a half-life of about one week — after a week, you have about half the level of the T4 still in your body, a week or so later you have half of that half remaining, and so on. Its full effects aren't reached until about six weeks after starting or changing a dose, which is why lab tests are optimally done every six weeks or so until a patient with hypothyroidism has reached satisfactory and stable thyroid hormone levels. T3, on the other hand, has a half-life of about a day. People on T3 sometimes feel its effects within minutes after taking it.

T3 is available as a separate synthetic medication with the brand name Cytomel in the US and Canada, and Tertroxin in the UK. It's usually prescribed along with a synthetic T4 medication. In the US, a synthetic T4/T3 combination is available with the brand name Thyrolar.

Natural, **desiccated thyroid** from pigs' thyroids with the brand name Armour is sold in the US, and in Canada, desiccated thyroid with the brand name Thyroid is made by Erfa (formerly by Pfizer and before that, by Parke-Davis). Westhroid and Nature-throid are available in the US, and Nature-throid is also available in Europe.

T3 or not T3

A thyroid gland that functions normally produces T4 and T3. Twenty percent of the T3 circulating in the body comes directly from the thyroid gland, and the remaining 80 percent comes from conversion of T4. Because of this conversion process,

T3 Files

T3 Supplementation
An explanation of the thyroid hormone T3 (triiodothyronine) and why supplementation of T3 along with T4 (thyroxine) is extremely beneficial to many people with hypothyroidism

T3 References
An overview of the references in this section

From medical journals and associations

1. T4/T3 Combination Therapy and Euthyroidism
Web page
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2. T4-to-T3 Conversion and Hypothyroidism
Web page
Printer-friendly

3. Hypothyroidism, T3, Mental Function, and Depression
Web page
Printer-friendly

4. Hypothyroidism, T3,

most doctors prescribe only synthetic T4 medication (Synthroid, Levoxyl, Levothroid, Eltroxin, Unithroid, and others). Many patients with hypothyroidism do fine on T4 only.

However, many others don't, and they need T3 supplementation in addition to T4. If the thyroid gland is malfunctioning and not producing enough — or any — T4, why assume that it still puts out enough T3, or that the body converts enough of its T4 to T3?

The addition of T3 often helps with many symptoms of hypothyroidism that may not disappear with supplemental T4 only. It has improved people's libido, memories, and vision. It has eliminated or greatly reduced brain fog, feeling cold, constipation, depression, chronic fatigue, headaches, insomnia, muscle and joint pain, and chronic sinus infections. For some people, but not all, it has helped them finally lose weight. A small percentage of people who try it feel worse or no better on it.

T3 tests

Do **lab tests** show if a person needs T3 supplementation? Sometimes. If free T3 is lower in its range than free T4 is, this suggests that more T3 would be beneficial for that person. On the other hand, some people who have posted in alt.support.thyroid have had lab results that did not indicate a problem with T3, but they still had symptoms of hypothyroidism, and the addition of T3 helped them.

Lab results do not tell the whole story. However, most people with hypothyroidism in alt.support.thyroid feel best when their free T4 and free T3 levels are in the upper part of their ranges. The exception is with people who are on desiccated thyroid. Because it contains a higher ratio of T3 to T4 than our thyroids produce, people taking it have a free T4 level that's lower in its range when the free T3 level is where it should be, in the upper part of its range.

Tell my doctor

Doctors are taught in medical school that T4 is the only thyroid medication that patients with hypothyroidism need. For many patients, that's true. The problem is that many other patients are left with reduced quality of life on T4 only — but their health improves greatly once T3 is added.

The medical establishment is increasingly looking at T3 in

and Heart Disease
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From other sources

1. From doctor-written articles
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2. From interviews with doctors
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3. From websites other than the above
Web page
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4. From books
Web page
Printer-friendly

Patients' Experiences

Printable compilations of patients' own words about how they felt after they started taking T3
Improved Life
Mental State
Symptoms

addition to T4 as essential treatment for some hypothyroid patients. Not having been educated on its use, however, some doctors are hesitant to prescribe it. That's why we've compiled **references** from medical journals and other sources that discuss the effectiveness, safety, and necessity of T3 supplementation for many patients with hypothyroidism.

To find a doctor who prescribes T3 for some patients, see our **tips on finding a good thyroid doctor**. To try to convince your current doctor to prescribe T3, you can bring medical references (see the column at the right) or send them to your doctor before your appointment and ask your doctor to prescribe at least a trial amount of T3.

Tell me more

See the links at the right on this and all the pages in the T3 section of this site. The **T3 References** page provides an overview of the references and why we need them. The compilations of references are divided into medical journal references and references from other sources, and each has a printer-friendly version so that you can take these references to your doctor if they're relevant to your situation.

The above list of symptoms that T3 has helped with is from the "T3: Patients' Experiences" compilations.

If you want to discuss T3 supplementation with your doctor, we recommend that you read through the files here, and perhaps print copies of them for your doctor. Because T3 is so much stronger and faster acting than T4, it's important to get the doses right. On pages 285 and 286 of his book *The Thyroid Solution*, Dr. Ridha Arem describes how to adjust T4 doses when supplementing T4 with T3. Some people in our group have brought this book to their doctors, and their doctors have used this protocol to successfully add T3 to the medication mix.

Kevin G. Rhoads discusses TSH, T3, and T4 in more detail in his **Thyroid 101 and Basic Fallacies** post. See also the related articles **The Desiccated Thyroid Story** and **The TSH Story** as well as **Hypothyroidism Medication** and **Hypothyroidism Medication Comparison**.

Lois Summers

This page was last updated June 14, 2008.

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

Application No.:	09/520,087
Confirmation No.:	1098
Filing Date:	March 7, 2000
Applicant:	Valerie Anne SCOTT et al.
Group Art Unit:	2859
Examiner:	Yaritza GUADALUPE
Title:	OPTICAL SIGHT
Attorney Docket:	10215-000022/US

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June 17, 2009

STATEMENT BY MR. HERMAN R. HEFLIN III

Sir:

In support of the Petition to Revive Under 37 CFR § 1.137(b) submitted concurrently herewith, please consider the following information.

1. On March 12, 2009, the Applicants' Swedish representative, Ström & Gulliksson AB ("S&G") instructed me, Herman R. Heflin III, to investigate the status of United States Application No. 09/520,087 ("the '087 application").

2. On March 13, 2009, and in accordance with instructions from S&G, I electronically filed (1) a general Power of Attorney (PTO/SB/80), which was provided by the Swedish representative, and (2) a Rule 3.73(b) Statement in the USPTO to associate the '087 application with my firm. The Power of Attorney was accepted by the USPTO via a Notice dated March 25, 2009.

3. Beginning March 25, 2009, I began to review the prosecution history of the '087 application using the USPTO's private PAIR system. The prosecution history of the '087 application was not available to me prior to March 25, 2009.

4. Between March 25, 2009, and June 4, 2009, S&G and I discussed the circumstances surrounding the delay in responding to the May 12, 2004 Office Action. Based on that correspondence, S&G sent instructions to me on June 5, 2009, for responding to the May 12, 2004 Office Action.

5. Between June 5 and June 17, 2009, I prepared the reply to the May 12, 2004 Office Action, the Petition to Revive Under 37 CFR § 1.137(b) and this Statement. In connection with preparing the Petition, I contacted Mr. Steven S. Payne directly regarding his Statement, which is submitted concurrently herewith.

6. I declare that all statements made herein of my own knowledge are true, and that all statements made on information and belief are believed to be true. These statements were made with knowledge that willful false statements and the like so made are punishable by fine or imprisonment, or both, under Section 1001 of Title 18 of the United States Code, and that such willful false statements may jeopardize the validity of the application or any patent issuing thereon.

Respectfully submitted,

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